ORIGINAL ARTICLE

Incidence of syncope and cardiac arrest in patients with severe aortic stenosis

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KEY WORDS

aortic stenosis, cardiac arrest, syncope

ABSTRACT

INTRODUCTION Syncope and sudden cardiac arrest are known complications of aortic stenosis (AS).

OBJECTIVES The aim of the study was to investigate the incidence of these complications in patients with severe symptomatic AS and to analyze whether basic clinical data and electrocardiographic (ECG) and echocardiographic parameters can be the markers of these complications.

PATIENTS AND METHODS The incidence of syncope and sudden cardiac arrest and its correlations with clinical and diagnostic data (ECG, echocardiography, Holter monitoring) were analyzed in 514 patients (mean age, 60 ±11 y) with severe symptomatic AS before valve replacement.

RESULTS Syncope was reported in 167 patients (32%), and aborted cardiac arrest in 14 (2.7%; ventricular fibrillation, 13 patients; third-degree atrioventricular block, 1 patient). None of the analyzed parameters was related to syncope. Patients with a history of sudden cardiac arrest had higher New York Heart Association class ($P = 0.01$), more frequent history of syncope ($P = 0.017$), higher left ventricular mass index ($P = 0.02$), lower ejection fraction ($P = 0.004$), longer QRS duration ($P = 0.048$), corrected QT ($P = 0.002$), QT dispersion ($P = 0.007$), and a higher number of ventricular arrhythmias in 24-hour Holter monitoring ($P = 0.002$). A multivariate analysis showed correlations between syncope, ejection fraction of less than 45%, and QTd exceeding 60 ms and aborted cardiac arrest. At least 2 of these parameters were observed in 8 of 14 patients ($P < 0.001$): sensitivity, 57%; specificity, 86%; positive predictive value, 10%; and negative predictive value, 98%.

CONCLUSIONS The incidence of sudden cardiac arrest in severe symptomatic AS is low. It is higher in patients with a history of syncope, prolongation of QTd, and reduced ejection fraction. None of the clinical and diagnostic parameters were associated with a history of syncope in patients with AS.

INTRODUCTION Aortic stenosis (AS) is the most common valvular heart disease in adults without explained pathogenesis and is regarded as the coming plague of the 21st century.[1-4] One of the axioms of clinical cardiology is that significant AS predisposes to syncope and also increases the risk of cardiac arrest.

The risk of syncope and cardiac arrest is associated with greater severity of valve defect, left ventricular dysfunction or hypertrophy, and coronary artery disease (CAD).[4-10] This widely accepted concept is based mainly on retrospective studies from the 1960s and 1970s, involving highly selected patients from reference centers. Usually, these were case reports of sudden cardiac death in patients with congenital or rheumatic valve disease.[5,7,11-16]

The aim of the study was to investigate the incidence of syncope and aborted cardiac arrest in patients with AS and to analyze whether simple clinical, electrocardiographic (ECG), and echocardiographic parameters can be the markers of these complications.

PATIENTS AND METHODS This was a retrospective study involving 514 consecutive patients with isolated AS before valve replacement (data collection, 1995–2008). AS was diagnosed on the basis of medical history, physical examination, and echocardiography. Patients with coexisting significant mitral or tricuspid valve disease or with moderate or severe aortic regurgitation were excluded from the study. All patients were symptomatic and fulfilled the echocardiographic criteria for

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TABLE 1 Baseline echocardiographic, electrocardiographic, and 24-hour Holter monitoring data in the study group

echocardiography		
LVMI, g/m²	220 ±66	91–514
EF, %	61 ±16	8–80
EF <45%	63 (12)	
MAG, mmHg	98 ±31	40–200
electrocardiogram		
heart rate, bpm	71 ±13	44–125
QRS, ms	102 ±19	65–184
QTc, ms	456 ±39	312–608
QTc >500 ms	65 (13)	
QTd, ms	61 ±24	20–180
Qtd >60 ms	158 (31)	
24-hour Holter monitoring		
heart rate, bpm	71 ±10	48–110
ventricular ectopy, n/24 h	484 ±1993	0–24,509
ventricular ectopy >300/24 h	95 (18)	
VT	59 (11)	
SVE, n/24 h	522 ±2326	0–42,466
SVT	246 (48)	

Data are presented as number (percentage) or mean ± standard deviation and ranges (min–max).

Abbreviations: LVMI – left ventricular mass index, MAG – maximal aortic gradient, QTc – corrected QT, QTd – QT dispersion, SVE – supraventricular ectopy, SVT – supraventricular tachycardia, VT – ventricular tachycardia

severe AS including: an aortic valve area, <1.0 cm²; mean aortic valve gradient, >40 mmHg; and aortic jet velocity, >4 m/s (all of the parameters or any combination thereof).[4] Data on prehospital medical treatment were collected: 232 patients received β-blockers; 134, angiotensin-converting enzyme inhibitors; 197, diuretics; 10, digoxin; 5, calcium channel blockers; 3, mexiletine; 2, sotalol; and 1, amiodarone. In 209 patients, no medications had been administered. All patients had normal serum electrolyte levels.

We recorded a history of syncope (excluding patients with a history of syncope not related to cardiovascular abnormalities, for example, neurologic syncope, etc.); a documented history of aborted cardiac arrest, paroxysmal atrial fibrillation (in ECG), and myocardial infarction; and New York Heart Association (NYHA) class.

Echocardiography We measured left ventricular end-diastolic diameter (LVEDd), end-systolic diameter, interventricular septum (IVS), posterior wall thickness (PWT), ejection fraction (EF), and maximal transvalvular systolic gradient. Aortic valve area was not analyzed because the measurement of that parameter was available in less than half of the patients. Left ventricular mass (LVM) was calculated and adjusted for the body surface area (BSA in m²) using the following formula:

$$LVMI\ [g/m^2] = 1.04[(IVS + LVEDd + PW)^3 - LVEDd^3] - 13.6/BSA.$$

Standard electrocardiogram We measured heart rhythm and rate, QRS duration, QT manually measured in all 12 leads (50 mm/s; corrected with the Bazet formula [QTc]), and QT dispersion (difference between maximal and minimal QT in 12 leads [QTd]).

24-hour Holter monitoring We measured the mean heart rate, the number of ventricular and supraventricular ectopies (VE and SVE, respectively), the presence/number of episodes of ventricular and supraventricular tachycardia (VT and SVT) (3 or more complexes, rate, >100 bpm).

Coronary angiography Coronary angiography was performed in all patients with indications. Significant CAD was defined as a reduction of at least 70% in the diameter of the major coronary artery or 50% in that of the left main coronary artery.

Statistical analysis The data were analyzed using SPSS 8.0. The continuous data were expressed as mean ± standard deviation, and the categorical data as percentage. The mean values of normally distributed variables were compared with the *t* test, and of the variables without normal distribution with the Wilcoxon test. The categorical values were analyzed with the χ^2 test. A univariate analysis and multivariate logistic regression were used to analyze correlations between syncope and aborted cardiac arrest and selected parameters. In a multivariate analysis, we included variables that were statistically significant in the univariate analysis; we used backward elimination with a *P* level of 0.1.

RESULTS The study population consisted of 197 women and 317 men (mean age, 60 ±11 y). Most patients were classified as NYHA class III (n = 222, 43.2%) or IV (n = 183, 35.6%). CAD was reported in 95 patients (18%), and a history of myocardial infarction in 40 (8%). The baseline characteristics of the patients are shown in TABLE 1.

The mean heart rate, QRS duration, and QTc and QTd were not correlated with medical treatment. Syncope was reported in 167 patients (32%) including 101 men and 66 women. None of the clinical and diagnostic parameters were associated with prior syncope in patients with AS (TABLE 2).

Documented cardiac arrest was reported in 14 patients (2.7%; 9 men and 5 women). Ventricular fibrillation was documented in 13 cases and third-degree atrioventricular block in 1. In 2 cases, cardiac arrest was recorded during 24-hour Holter monitoring (FIGURE 1). Patients with and without a history of cardiac arrest did not differ in terms of age. Patients with a history of cardiac arrest had a higher NYHA class ($P = 0.01$), more frequent history of syncope ($P = 0.017$), greater LVMI ($P = 0.02$), lower EF ($P = 0.004$), longer QRS ($P = 0.048$), longer QTc ($P = 0.002$), more extended QTd ($P = 0.007$), and more frequent ventricular ectopy during 24-hour Holter

TABLE 2 Comparison of the results between patients with and without a history of syncope

Parameter	Syncope	
	yes (n = 167)	no (n = 347)
clinical data		
age, y	59 ±11	60 ±10
sex, men/women	101/66	216/131
history of palpitations	115 (69)	212 (61)
paroxysmal atrial fibrillation	14 (8)	33 (9)
myocardial infarction	16 (9.6)	24 (7)
NYHA	3.1 ±0.77	3.1 ±0.7
echocardiography		
LVMI, g/m^2	217 ±66	221 ±67
EF, %	63 ±14	61 ±16
MAG, mmHg	101 ±31	96 ±30
CAD	22 (13)	73 (21)
electrocardiogram		
heart rate, bpm	70 ±12	71 ±13
QRS, ms	101 ±21	102 ±17
QTc, ms	452 ±37	458 ±39
QTd, ms	61 ±21	61 ±25
24-hour Holter monitoring		
heart rate, bpm	71 ±10	70 ±11
ventricular ectopy, n/24 h	163 ±364	638 ±2398
VT	15 (9)	44 (13)
SVE, n/24 h	565 ±1688	500 ±2581
SVT	80 (48)	166 (48)

Data are presented as number (percentage) or mean ± standard deviation.

Abbreviations: CAD – coronary artery disease, others – see TABLE 1

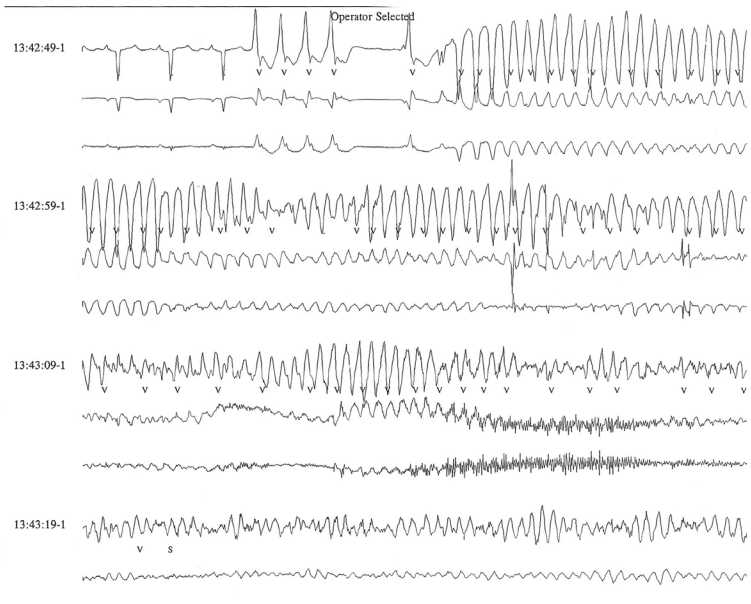

FIGURE The final part of continuous electrocardiogram (ECG) tracing in a 62-year-old woman with severe symptomatic aortic stenosis (maximal aortic gradient, 73 mmHg; ejection fraction, 27%; New York Heart Association class, IV; QTd, 80 ms; no history of syncope). The patient was resuscitated, underwent a surgery, and survived at least 12 years. The ECG shows sinus rhythm and then short run of VT; after a short pause, ventricular escape beat of the same morphology; then, premature ventricular beat of different morphology that initiates ventricular flutter; and then, ventricular fibrillation.

monitoring (P = 0.002) (TABLE 3). In the multivariate analysis, the incidence of cardiac arrest was independently correlated with a history of syncope (odds ratio [OR], 4.9 [1.5–16.0]), lower EF (0.96 [0.94–0.99]), and longer QTd (1.03, 1.009–1.051) (TABLE 4). We also observed a borderline correlation with the NYHA class. Medical treatment had no effect on the results. In another model, we used the same set of parameters but with arbitrary cut-off values. They were as follows: history of syncope; EF, <45%; QTd, >60 ms; QTc, >500 ms; QRS, >120 ms; ventricular ectopy, >300/24 h). We observed that EF, a history of syncope, and QTd were independently correlated with the incidence of aborted cardiac arrest (TABLES 4 and 5). At least 2 of these parameters were observed in 8 of 14 patients (57%) with cardiac arrest and in 70 patients (14%) without cardiac arrest (P <0.001; sensitivity, 57%; specificity, 86%; positive predictive value, 10%; negative predictive value, 98%).

DISCUSSION Numerous prospective studies assessed the risk factors of sudden cardiac death in different groups of patients, which in fact assessed the risk of arrhythmia.[17,18] In patients with severe symptomatic AS, this type of analysis is not possible because as soon as the patient becomes symptomatic, aortic valve replacement should be promptly performed.[4] In 1968, Ross and Braunwald[5] summarized the results of the retrospective studies examining the natural history of AS and evaluated the mean survival of patients following the onset of symptoms. The mean survival did not exceed 2 to 5 years in patients who developed symptoms of heart failure, angina, or syncope.

Owing to the lack of prospective studies, we decided to evaluate the history of syncope and cardiac arrest in patients with significant AS. Our patients constituted the biggest group analyzed so far (514 patients). We evaluated simple parameters, available in all patients before surgery.

The typical symptom of AS is syncope, which initially develops only during exercise or with changes of the body position. It is caused by decreased cerebral blood flow, also as a result of arrhythmias.[4,5,16,19-22] Left ventricular hypertrophy (present in patients with AS) and reduced EF are well known predictors of higher incidence of arrhythmias.[1,3,5,7,16,22-27] Conduction disturbances caused by calcifications are also possible. The prognosis becomes worse after the first episode of syncope.[5] In our study group, 167 patients (32%) experienced at least 1 episode of syncope. We did not find any specific parameter that distinguished between the group with and without syncope.

Documented aborted cardiac arrest was reported in 14 patients (2.7%). In the univariate analysis, the incidence of cardiac arrest was correlated with syncope, NYHA class, higher LVMI, lower EF, longer QRS, QTc, and QTd duration, and more frequent ventricular ectopy. In the multivariate

TABLE 3 Comparison of the results between patients with and without a history of aborted cardiac arrest

	Aborted cardiac arrest		*P* value
	yes (n = 14)	no (n = 500)	
clinical data			
age, y	62 ±14	60 ±11	
sex, men/women	9/5	307/192	
history of palpitations	9 (64)	157 (31)	0.017
paroxysmal atrial fibrillation	11 (79)	314 (63)	
myocardial infarction	2 (14)	45 (9)	
NYHA	3 (21)	37 (7)	
age, y	3.6 ±0.6	3.1 ±0.7	0.01
echocardiography			
LVMI, g/m^2	260 ±78	219 ±66	0.02
EF, %	50 ±20	62 ±15	0.004
EF <45%	7 (50)	56 (11)	0.001
MAG, mmHg	94 ±27	98 ±31	
CAD	4 (29)	91 (18)	
electrocardiogram			
heart rate, bpm	74 ±12	71 ±12	
QRS, ms	111 ±25	101 ±18	0.048
QTc, ms	489 ±48	455 ±38	0.002
QTc >500 ms	5 (36)	60 (12)	0.02
QTd, ms	79 ±30	61 ±24	0.007
QTd >60 ms	9 (64)	149 (30)	0.01
24-hour Holter monitoring			
heart rate, bpm	71 ±11	71 ±11	
ventricular ectopy, n/24 h	1093 ±2740	467 ±1971	0.002
VT	3 (21)	56 (11)	
SVE, n/24 h	570 ±1716	521 ±2344	
SVT	6 (43)	240 (48)	

Data are presented as number (percentage) or mean ± standard deviation.

Abbreviations: see TABLES 1 and 2

TABLE 4 Factors associated with the incidence of aborted cardiac arrest: the multivariate analysis

	Wald statistics	*P* value	Odds ratio (confidence interval)
QTd	7.6	0.006	1.03 (1.009–1.051)
syncope	6.9	0.008	4.9 (1.5–16.0)
EF	4.1	0.04	0.96 (0.94–0.99)
NYHA	2.6	0.1	2.29 (0.8–6.2)
effect of dychotomic parameters			
EF <45%	15.3	0.0001	10.4 (3.2–33.8)
syncope	8.2	0.004	5.8 (1.7–19.5)
QTd >60 ms	6.3	0.01	4.4 (1.4–14.1)

Abbreviations: NYHA – New York Heart Association, others – see TABLE 1

analysis, we observed a significant effect of syncope (5-fold higher risk), lower EF, and increased QTd on the risk of cardiac arrest. The NYHA class hshowed only a borderline correlation with the risk of cardiac arrest. Our findings partially agree with the previous studies. In most cases, the analysis of sudden cardiac death in patients with AS was retrospective and had been published from 10 to 20 years ago. The authors of all those reports emphasized that the risk was higher by 5% to 34% and rarely occurred in asymptomatic patients.[6,10,11,22] It has been suggested that significant left ventricular hypertrophy, high transvalvular gradient, critical CAD, and significant myocardial fibrosis were associated with an increased incidence of cardiac arrest; however, standard clinical and echocardiographic parameters were not useful in identifying patients at risk.

From 1958 to 1978, a total of 35 cases of sudden cardiac death in patients with AS were reported.[5,6,8-10,12,14,16,28-32] Of those patients, 75% were symptomatic, most had ST changes, and stenosis was severe in all autopsy-confirmed cases. There have also been reports of sudden cardiac death recorded in patients with AS during 24-hour Holter monitoring. Von Olhausen[7] described 7 patients; in 6 cases, the cause of death was malignant VT and only 1 death was associated with bradyarrhythmia. What is more, in the last hours before death, the frequency and complexity of ventricular ectopy and heart rate increased. In another study, Von Olhausen[31] also reported sudden death of a patient with severe AS and impaired left ventricular function (VF during 24-hour Holter monitoring), without prior exertion, syncope, and without any ventricular ectopy within the last hour.[31] Dilaveris et al.[33] described a case of a 77-year-old woman with asymptomatic (only palpitation related to atrial fibrillation) and moderate AS during Holter monitoring due to torsade de pointes, who died in sleep without prior arrhythmia. Sudden cardiac death was also documented on Holter monitoring in a 71-year-old man with significant AS by Siostrzonek et al.[28] The tracing showed the development of stress-induced sinus tachycardia with broad QRS and rapid transition to ventricular fibrillation. This is particularly interesting because the patient had no impaired left ventricular function.

The analysis of the Framingham study demonstrated that patients with hypertrophy had several times higher incidednce of ventricular arrhythmias and an increased risk of cardiac arrest.[34] The substrate for the increased number of ventricular ectopy was explained by the increase of the amount of connective tissue and myocardial perfusion abnormalities. Bikkina et al.[35] showed that left ventricular hypertrophy in patients with asymptomatic CAD and ventricular arrhythmia doubles the risk of death. In our group, the comparison of an average LVMI showed a significantly higher hypertrophy in patients with a history of cardiac arrest compared with those without such history.

In our study, increased frequency of ventricular ectopy was observed in patients with a history of cardiac arrest; however, this was only significant in the univariate analysis.

TABLE 5 Distribution of the number of risk factors in the analyzed groups

Risk factors	Cardiac arrest	
	yes (n = 14)	no (n = 500)
0	0	210 (42)
1	6 (43)	220 (44)
2	5 (36)	68 (14)
3	3 (21)	2 (0.4)

Data are presented as number (percentage).

In contrast to previously cited reports, we performed a more complex analysis of ECG findings, and we also analyzed repolarization. Progressive prolongation of the action potential duration and increased heterogeneity of ventricular repolarization are commonly found in hyperthrophied myocardium in patients with AS and may provide the substrate for the development of arrhythmias arising from triggered activity.[25,26,36] Left ventricular hypertrophy led to increased QTd and the development of arrhythmias.[23,36-38] The findings of Kosar et al.[23] demonstrated that maximum QTc and QTd were increased in patients with AS when compared with healthy subjects, and were also significantly greater in those with a history of syncope. In our previous study, we also confirmed increased repolarization parameters in patients with AS.[39] In the present study in patients with sudden cardiac arrest, we observed more frequent repolarization abnormalities expressed as prolonged QTc and QTd. In the multivariate analysis, we observed increased incidence of cardiac arrest with QTd prolongation, especially when QTd exceeded 60 ms. This finding clearly shows that the instability of repolarization seems to be more important than, for example, the presence of ventricular ectopy.

Several observational studies indicated that reduced EF can predict both sudden and nonsudden death.[3,5,7,19,22-28,40] The history of cardiac arrest in our group of patients with AS was higher in those with an EF of less than 45%.

From the clinical point of view, we observed that 3 simple parameters: an EF of less than 45%, a history of syncope, and QTd exceeding 60 ms, were independently correlated with the history of cardiac arrest. Cardiac arrest was reported 5.8-fold more often in patients with a history of syncope, 10-fold more often in those with an EF of less than 45%, and 4-fold in those with QTd prolongation exceeding 60 ms.

Study limitations Our study has several limitations. First, this was a retrospective study, but it should be noted that it is not possible to conduct a prospective observational study in patients with symptomatic AS. Second, we collected our data in consecutive patients who were referred to our department with symptomatic AS. Thus, our study group comprised patients from a reference center, and patients with less clinically complicated AS might have been referred to other hospitals. Third, we should stress that the incidence of sudden cardiac arrest was assessed based on a population of patients with AS who had survived cardiac arrest. Fourth, our analysis of arrhythmia was based only on 1 24-hour ECG; reproducibility of ventricular ectopy, especially VT is rather poor so this might have also affected our results. However, our group of patients with severe symptomatic AS is the largest such group in the literature.

One could also question the measurement of QTd, which is rarely used in clinical practice. We simply documented the potential value of QTd, which is automatically calculated by some ECG machines.

We did not find any statistically significant effect of medical treatment on ECG results; however, this should be considered in any ECG data.

In conclusion, the history of sudden cardiac arrest in patients with severe symptomatic AS is rather low. The incidence of cardiac arrest is higher in patients with a history of syncope, prolongation of QTd above 60 ms and a reduced EF of less than 45%. None of the clinical and diagnostic parameters were associated with a history of syncope in patients with AS.

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ARTYKUŁ ORYGINALNY

Występowanie omdleń i nagłego zatrzymania krążenia u pacjentów z istotnym zwężeniem zastawki aortalnej

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nagłe zatrzymanie krążenia, omdlenie, zwężenie zastawki aortalnej

STRESZCZENIE

WPROWADZENIE Omdlenia i nagłe zatrzymanie krążenia (NZK) to znane powikłania zwężenia zastawki aortalnej (*aortic stenosis* – AS).

CELE Celem badania była ocena występowania tych powikłań u pacjentów z ostrym objawowym AS oraz analiza czy podstawowe dane kliniczne oraz wyniki EKG i echokardiografii mogą być zwiastunami ich występowania.

PACJENCI I METODY Oceniono występowanie omdleń i NZK oraz ich związek z danymi klinicznymi i wynikami badań (EKG, echokardiografia, EKG rejestrowane metodą Holtera) u 514 pacjentów (średnia wieku 60 ±11 lat) z ciężkim objawowym AS przed wymianą zastawki.

WYNIKI Omdlenia wystąpiły u 167 (32%) pacjentów, NZK u 14 (2,7%) pacjentów (migotanie komór u 13, blok przedsionkowo-komorowy III° u jednego). Żaden z analizowanych parametrów klinicznych nie był związany z występowaniem omdleń. U pacjentów z NZK w wywiadzie stwierdzono wyższą klasę NYHA ($p = 0,01$), częstsze występowanie omdleń w wywiadzie ($p = 0,017$), wyższy indeks masy lewej komory ($p = 0,02$), niższą frakcję wyrzutową ($p = 0,004$), dłuższy czas QRS ($p = 0,048$), QTc ($p = 0,002$), dyspersję QT ($p = 0,007$) i większą liczbę pobudzeń komorowych w EKG rejestrowanym metodą Holtera ($p = 0,002$). Analiza wieloczynnikowa wykazała korelacje między występowaniem omdleń, EF <45% oraz dyspersją QT >60 ms a NZK w wywiadzie. Przynajmniej dwa z tych parametrów rejestrowano u 8 z 14 pacjentów z NZK w wywiadzie ($p < 0,001$): czułość 57%, swoistość 86%, wartość predykcyjna wyniku dodatniego 10%, wartość predykcyjna wyniku ujemnego 98%.

WNIOSKI U pacjentów z ciężkim objawowym AS częstość występowania NZK w wywiadzie jest niska. Jest ona wyższa u pacjentów, u których stwierdzono omdlenia w wywiadzie, wydłużoną dyspersję QT oraz obniżoną frakcja wyrzutową. Nie znaleziono związku między wynikami badań a występowaniem omdleń w grupie pacjentów z AS.

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